

REMARKS

After entry of this amendment, claims 1-3, 6-17, 19-20, 23, and 25-27 are pending, of which claims 12 and 13 are withdrawn. Claims 22 and 24 have been cancelled without prejudice or disclaimer, and the subject matter has been incorporated into the amended claims 1 and 14, respectively. New claims 26 and 27 have been added and find support in the original claims 6 and 15, respectively. The claims have been amended without prejudice or disclaimer and find support *inter alia* in the original claims. Further support for the amended claims 1 and 14 is found in the specification at page 55, line 40. Claim 25 has been amended without prejudice or disclaimer to correct a typographic error. No new matter has been added.

Claim Rejection – 35 U.S.C. § 112

Claims 1-3, 6-11, 14-17 and 19-20 were rejected under 35 U.S.C. § 112, first paragraph, for lack of an enabling disclosure. Applicants respectfully disagree. However, to expedite prosecution, the claims have been amended without prejudice or disclaimer to recite the Bax inhibitor-1 (“BI1”) as being a protein sharing a sequence identity of at least 90% at the amino acid level with the sequence of SEQ ID NO: 2. Applicants respectfully submit that the claims as amended recite a scope of subject matter which a skilled artisan could clearly make and use according to the teaching in the specification. This is further evidenced by the fact that claims 22-25 were not included in this rejection in the Office Action dated February 7, 2008. Accordingly, reconsideration and withdrawal of this rejection is respectfully requested in view of the present amendment.

Claim Rejection – 35 U.S.C. § 102(a)

Claims 1-2, 6-11, 14-17 and 19-20 were rejected under 35 U.S.C. § 102(a) as being anticipated by Simmons *et al.* (WO 02/101079, hereinafter “Simmons”). Applicants respectfully disagree. However, to expedite prosecution, the claims have been amended without prejudice or disclaimer. It is believed that this rejection is rendered moot in view of the present amendment. Reconsideration and withdrawal of the rejection is respectfully requested.

Claim Rejection – 35 U.S.C. § 103(a)

Claims 1-3, 6-11, 14-17, 19-20, and 22-25 were rejected under 35 U.S.C. § 103(a) as being obvious over Simmons, in view of Hückelhoven *et al.* (Plant Mol. Biol., 2001, 47: 739-

748, hereinafter "Hückelhoven"). Applicants respectfully disagree and traverse the rejection for the following reasons.

To support a *prima facie* conclusion of obviousness, the prior art must disclose or suggest all the limitations of the claimed invention. See *In re Lowry*, 32 F.3d 1579, 1582, 32 USPQ2d 1031, 1034 (Fed. Cir. 1994).

As disclosed in the specification at page 4, lines 23-36, the present invention provides an efficient defense method without breaking any existing resistance to other pathogens (e.g., mlo-mediated resistance). To achieve such a result, it is found to be important to have the expression of the B11 protein remained **essentially unchanged or reduced in the leaf epidermis** of a plant while the amount or the function of the B11 protein is increased in at least one plant tissue other than the leaf epidermis of the same plant.

Simmons discloses BI genes from corn and soybean, and methods of using these sequences in generating transgenic plants and in improving disease and stress resistance. Although Simmons teaches tissue-specific promoters such as root-, seed- or flower-specific promoter may be used to over-express BI genes, Simmons does not teach or suggest that the expression of the B11 protein should remain essentially unchanged or reduced in the leaf epidermis of the transgenic plants as required by the claimed method. This is evidenced by the disclosure throughout the specification of Simmons. For instance, in Example 11, Simmons suggests that the BI genes could be used in crop plants to retard cell death and senescence. Simmons further suggests that tissue-preferred promoters, especially promoters specific to the tissues most accounting for ear mold ingress, namely ears, husk, pericarp or cob, could be used to drive the expression of the BI genes in transgenic plants. See Simmons at pages 67-68. Simmons, however, does not teach or suggest in any way that the expression of the BI genes in the transgenic plants should remain essentially unchanged or reduced in the leaf epidermis to avoid breaking any existing resistance to other pathogens.

The disclosure of Hückelhoven does not remedy this deficiency. Hückelhoven teaches a nucleic acid encoding a B11 protein that is 100% identical to SEQ ID NO: 2. It is shown that the expression of this barley B11 gene was up-regulated in barley leaves in response to *Blumeria graminis* f.sp. *hordei* (Bgh) inoculation. Hückelhoven, page 743, right Col. and page 745, Figure

5. Hückelhoven, however, does not in any way teach or suggest that the expression of this BI1 gene is essentially unchanged or reduced in the barley leaf epidermis.

It follows that the combination of Simmons and Hückelhoven does not render the present invention obvious because the combined teaching does not teach or suggest that the essentially unchanged or reduced expression in the leaf epidermis of the BI1 protein plays an important role in the successful increase of resistance as achieved by the claimed method.

Even assuming *arguendo* that it would have been routine for a skilled artisan to perform an expression analysis to arrive to an expression profile of the BI1 gene, it would not have been obvious to a person of ordinary skill in the art to realize the importance of maintaining the expression of the BI1 protein essentially unchanged or reduced in the leaf epidermis of a transgenic plant simply from a routine expression profiling experiment.

The Examiner further alleges that, given the teaching of Simmons and Hückelhoven, one skilled in the art would expect that expression of the barley BI1 gene in a transgenic plant would increase or induce resistance against any necrotrophic or hemibiotrophic plant fungal pathogen. Applicants respectfully disagree.

As shown in Comparative Example 1 at pages 92-95 and Figure 10(A) of the specification, over-expressing the BI1 gene in epidermis confers an increase of *Bgh* infection (*i.e.* susceptibility, as represented by penetration efficiency) rather than the alleged increase of resistance to *Bgh* infection. Only when the expression of the BI1 gene in the epidermis is essentially unchanged or reduced (*e.g.*, by antisense suppression), a decrease of *Bgh* infection (*i.e.* increased resistance to *Bgh*) is observed (see Figure 10(B)). Because neither Simmons nor Hückelhoven, or the combination thereof, teaches over-expressing a BI1 gene in a transgenic plant with the expression of the BI1 gene essentially unchanged or reduced in the leaf epidermis of the plant to generate or increase resistance to biotic or abiotic stress factor, the references cited by the Examiner, alone or in combination, do not render the claimed method obvious. Reconsideration and withdrawal of this rejection is respectfully requested.

Separate consideration to the newly added claims 26 and 27 is respectfully requested. It is noted that neither Simmons nor Hückelhoven teaches the use of a tuber- or mesophyll-specific promoter.

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